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Reduction of human sleep duration after bright light exposure in the morning

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In 8 subjects the spontaneous termination of sleep was determined after repetitive exposure to either bright or dim light, between 6 00 and 9 00 h, on 3 days preceding sleep assessment. Sleep duration was significantly shorter following bright light than following dim light. During sleep the time course of EEG energy was not affected by the light treatment. Analysis of the time course of body temperature during sleep indicated an earlier rise of body temperature following the bright light treatment. In terms of the two-process model of sleep regulation this can be interpreted as a direct effect of light on the circadian phase of the wake up threshold.

The duration of human sleep is determined by both homeostatic and circadian factors. A homeostatic component has been demonstrated in experiments in which sleep debt at a fixed sleep onset time was manipulated by varying the length of the preceding sleep episode. With increasing sleep debt an increase in sleep duration was observed [2, 17]. The changes in sleep duration are small and not proportional to the variations in sleep debt [10]. This may be explained by postulating that sleep has an intensity dimension. Electroencephalogram (EEG) studies revealed that the amount of slow-wave sleep increases with increasing duration of prior wakefulness [16]. Furthermore, spectral analysis of the sleep EEG showed that after sleep deprivation EEG power density increases within all sleep stages [4]. So, the homeostatic aspects of sleep regulation are not limited to sleep duration but also encompass changes within sleep.

The circadian influence on sleep duration has been inferred from experiments in which the circadian phase of sleep onset was varied. If this was achieved by extending the duration of prior wakefulness, contrary to the predictions from a simple homeostatic model, sleep duration decreased with increasing duration of prior wakefulness up to about 32 h [1]. The circadian influence on sleep duration is also present under

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conditions of temporal isolation. Longest sleep episodes occur when sleep is initiated near the maximum of the body temperature rhythm whereas shortest sleep episodes start near the minimum of the body temperature rhythm [6, 20].

In the two-process model of sleep regulation [5, 9] both the homeostatic and the circadian aspects of sleep duration are accounted for. During wakefulness a regulatory variable (S) increases until an upper threshold is reached and sleep is initiated. During sleep S decays exponentially until a lower threshold is reached. This results in the transition from sleep to wakefulness. The time course of S is reflected in the EEG power density (0.25–15.0 Hz) during sleep. Under normal conditions waking up occurs on the rising part of the lower threshold which coincides with the rising part of the body temperature curve. The two thresholds are modulated over the circadian cycle. This results in variation of sleep duration with the phase of sleep onset. The threshold variations are thought to be generated by a circadian pacemaker, presumably located in the suprachiasmatic nuclei. The period of the threshold rhythm is identical to the period of the body temperature rhythm, which is approximately 25 h in the absence of Zeitgebers [19].

Under natural conditions circadian rhythms are synchronized to Zeitgeber cycles with a period of 24 h. From animal studies it was concluded that the light–dark cycle is a powerful Zeitgeber [3]. An essential feature of the process of entrainment is the phase-dependent sensitivity of the circadian system to light. For example, in the diurnal squirrel monkey, light pulses given just prior to the activity onset phase advance the drinking rhythm, whereas phase delays can be induced by light pulses at the end of the activity period [11].

In man the role of light in the process of entrainment has been debated and social factors have long been thought to be more powerful Zeitgebers [18]. However, experiments in which the imposed light–dark cycle was adequately controlled showed that also the human circadian system can be entrained by light–dark cycles [7]. There is a controversy on the mechanism by which light exerts its influence on the circadian system. In the model of Kronauer and colleagues the effects of light are mediated by shifts of the sleep–wake cycle [12]. In the two-process model light acts directly on the circadian pacemaker, which in turn exerts its control over the sleep–wake cycle. The present experiment was designed to differentiate between these two alternatives.

In February and March of 1986, 8 male subjects (age 23.1 ± 2.5 (S.D.) years) participated in a cross-over design of exposure to two light conditions. In both instances they came to the laboratory on 4 consecutive evenings. During the first 3 evenings they were sitting from 19.00 to 22.00 h in a darkened room at a light intensity of 1 lux. From 22.00 till 06.00 h they were allowed to sleep in a completely dark room. Between 06.00–09.00 h, they were sitting awake in a laboratory room. In this room light intensity was kept at 1 lux (candle light) in one condition whereas under the other condition light intensity was increased to 2000 lux by white fluorescent tubes (vita lux). Four subjects entered the bright light condition first and were subjected to the dim light treatment 3 weeks later. In the other 4 subjects the order was reversed.

On the fourth evening of both conditions subjects were sitting in a darkened (1

lux) room from 18.00 until 24.00 h when sleep was allowed to start. The subjects were instructed not to rise until they felt refreshed. They slept in a darkened room and had no knowledge of clock time. During this experimental night body temperature, electromyogram (EMG), electrooculogram (EOG) and EEG were recorded. The EEG was derived from C₃-A₂ and C₄-A₁. Paper recordings were made at a paper speed of 10 mm s⁻¹ and were scored according to the criteria of Rechtschaffen and Kales [15]. After low pass filtering at 25 Hz (24 dB/oct) the EEG was digitized with a sampling rate of 64 Hz. EEG power densities between 0.25–15.0 Hz were calculated per 4-s periods, by means of a fast Fourier transformation. For an estimation of the time course of *S* the accumulation of EEG energy (0.25–15.0 Hz) during sleep within stages 1, 2, 3, 4 and R.E.M. sleep was calculated. Sleep onset was defined as the first occurrence of stage 2, provided that less than 2 min of stage 0 (waking) or 1 was present in the next 10 min.

The average time of sleep onset was virtually identical for both conditions: 00.24 h \pm 2.5 min (S.E.M.), after exposure to darkness vs 00.20 h \pm 2.9 min (S.E.M.) after exposure to bright light. Sleep latencies in the two conditions did not differ either: 19 \pm 3.5 (S.E.M.) min vs 15 \pm 1.9 (S.E.M.) min after dim and bright light, respectively. For the determination of the effects of treatment on the spontaneous termination of sleep we proceeded in two ways. First the end of sleep was operationally defined as the beginning of the first 15-min interval after sleep onset in which no epoch of stage 2, 3, 4 or R.E.M. sleep was present. By this definition of sleep end the resulting clock times were 08.42 h \pm 18.2 (S.E.M.) min for the dark condition vs 07.44 h \pm 7.44 (S.E.M.) min for the light condition ($P < 0.05$; Wilcoxon matched-pairs signed rank test). The resulting sleep durations were 498.6 \pm 18.6 (S.E.M.) min vs 444.0 \pm 10.9 (S.E.M.) min for the dark and light condition respectively ($P < 0.05$, Wilcoxon matched-pairs signed rank test).

For a less arbitrary evaluation of waking up tendency, the accumulation of stages 0, 1 and movement time after sleep onset was calculated and plotted at 30-min intervals. Fig. 1 shows that the curves for both conditions are identical during the first hours after sleep onset, but dissociate after 7 h. Wakefulness accumulates faster in the night after the light treatment.

In the two-process model a shortening of sleep may result from either a lower level of *S* at sleep onset, a faster decay of *S* during sleep or a change of the wake-up threshold at sleep end. Fig. 1 therefore depicts the accumulation of EEG energy during the two nights. The data are expressed relative to the amount of energy accumulated during the first 6 h of the night after the dim treatment (= 100%).

During the first 6 h of sleep the two curves are virtually identical. In the first 6 h of the night after the bright light treatment the amount of energy accumulated (98.22 \pm 4.8% (S.E.M.)) was not significantly different from 100%. The absence of a difference in both the amount of EEG energy accumulated and its time course indicates identical levels of *S* at sleep onset and identical decay rates during sleep under the two conditions.

The shortening of sleep then may be explained by a change in the wake-up threshold. Although a physiological correlate of this threshold remains to be identified,

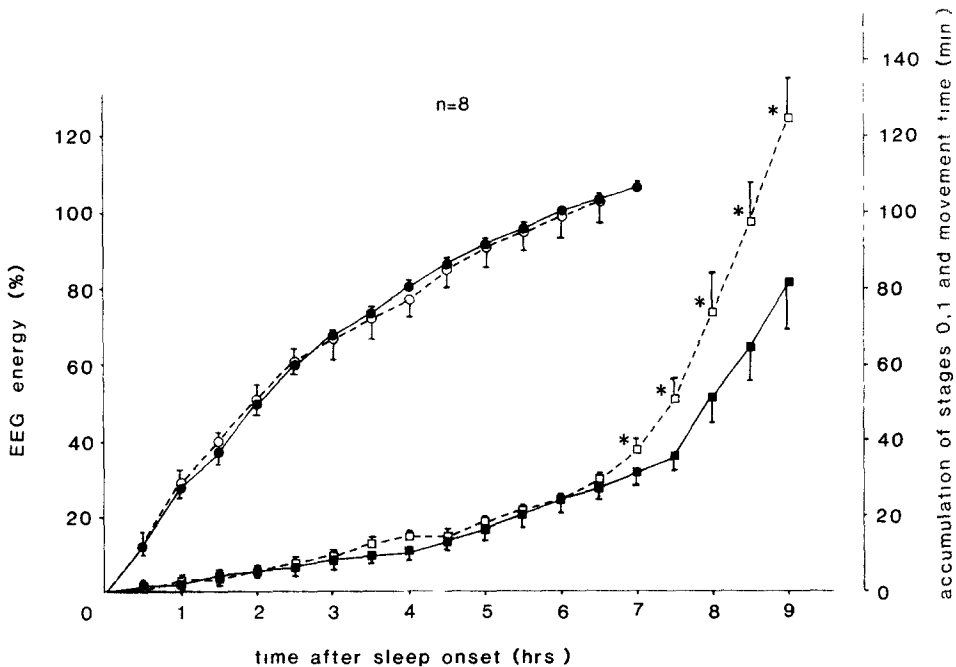


Fig. 1 Rectangles: accumulation of stages 0, 1 and movement time (\pm S.E.M.). * $P < 0.05$, Wilcoxon matched pairs signed ranks test. Circles: accumulation of EEG energy (0.25–15.0 Hz) within stages 1, 2, 3, 4 and R.E.M. sleep during the night after the dim light treatment and after the bright light treatment. Bars indicate 1 S.E.M. 100% = EEG energy accumulated during the first 6 h of the night after exposure to dim light. Filled symbols, after dim light, open symbols, after bright light.

the time course of body temperature may provide some information. Due to technical difficulties body temperature recordings for both conditions were obtained in only 6 subjects.

The 10-min interval with the lowest median body temperature value was located at 02.30 h \pm 49.8 (S.E.M.) min after the dim light condition vs 01.30 h \pm 17.0 (S.E.M.) min after the light treatment. The difference was not statistically significant. It should be kept in mind though that the minimum of body temperature is not a precise marker of the endogenous rhythm.

For a further analysis of the time course of body temperature the average temperatures per 30 min were expressed as deviations from the mean temperature between 00.00 and 07.00 h (Fig. 2). Analysis of variance revealed that the difference between the two conditions was significant ($F_{13,65} = 2.71$; $P < 0.01$). The difference can be interpreted as an earlier rise of body temperature after the bright light treatment as compared to the time course of body temperature after exposure to dim morning light.

In conclusion, repetitive treatment with bright light in the early morning advanced wake up time relative to wake up time after exposure to dim light. Since during both treatments sleep was scheduled at the same clock times, this effect must be attributed to an effect of light *not* mediated through the sleep wake behaviour. This conclusion

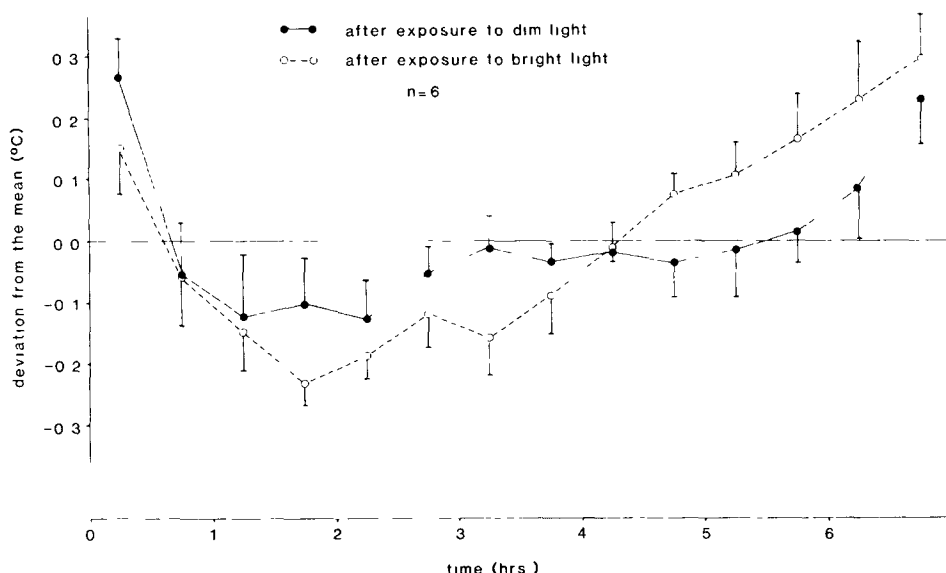


Fig. 2 Time course of rectal temperature during the night after exposure to dim light and after exposure to bright light. Data are expressed as deviations from the mean temperature between 00.00 and 07.00 h. Bars indicate 1 S.E.M.

is in agreement with a recent experiment of Czeisler et al. [8] in which in one subject, exposure to bright light in the evening, while the sleep-wake cycle was fixated, induced a delay of the circadian rhythms in body temperature and cortisol secretion. Since the time course of EEG energy was not different after the two treatments it must be concluded that the dynamics of process *S* was not affected. The explanation that sleep after light treatment is shorter because of a change in the wake up threshold is supported by the difference in time course of body temperature after the two treatments.

When animals living in constant conditions are exposed to bright light pulses, their major reaction is a phase shift of their circadian rhythms. It is tempting, therefore, to assume a similar shift to occur in the human wake-up threshold subsequent to morning light treatment. The direction of the shift following bright light is consistent with the advance shift observed in circadian rhythms of animals exposed to light in their late subjective night and is indeed a theoretical prerequisite for entrainment by light [14].

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